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EDITORIAL

ALTHOUGH it is now well established that miliary tuberculosis can run a chronic course, or even heal, we are poorly informed upon many aspects of its prognosis. There is, for instance, no statement in the literature of the relative frequency of the acute and chronic forms, nor of the proportion these bear to one another in differing racial stocks, at different ages, or under varying social and economic circumstances. While the acute variety is certainly by far the commoner, whatever the soil in which it happens to arise or whatever the associations with which it is bound, we still lack any precise expression of this general opinion. That the chronic form is a rarity is not enough excuse, for there is a pretty general opinion that it is by no means so great a rarity as was believed some years ago. Most of those who see a good deal of pulmonary tuberculosis come across a few cases each year; and the lack of adequate figures may well be due to the fact that many of these cases are never placed on record, rather than to a rarity which this very absence of record serves to keep fictitiously alive.

Certainly then we need to learn much more upon the prognosis of these miliary forms of the disease. One review of the chronic cases some years ago, comprising 64 examples, showed that of these 13 died within six months, a further 33 eventually, leaving only 18 in whom the disease became arrested for an average period of observation of seven years. The fatal rôle of meningitis as the leading cause of death, the frequency of a relapsing course among those who live for more than six months but who die eventually, the deceptiveness of remissions within two years of onset and yet the permanence of recovery for a small number after this length of time, were the chief features of this particular survey of the prognosis from the collected record of the time. Little has been added since then, but Illing now reports another 21 cases, all children. Although the length of observation with some of these has not been very long, several points of unusual interest emerge.

Dealing as he was with children in whom it is generally reckoned that miliary tuberculosis is at its most dangerous zenith, he found that in no less than 12 of his 21 chronic cases the lesions retrogressed or resolved as judged by serial radiographs. In nine the miliary opacities gradually disappeared entirely, and this within a year of the time they were first detected. That such a favourable experience should have been the lot of a single observer is indeed remarkable; but it shows, at least, that the child is not exempt from some capacity for recovery that may rival that of the adult. Thirteen of his 21 cases are still alive, 11 of these after more than a year from the onset and 6 more than two years.

Combining his own series with 32 others from the literature, Illing found that in these 53 children with chronic miliary tuberculosis there was no special tendency for survival to be restricted to the older age groups. Nor, to judge from his figures, is the incidence of the chronic form unduly slight in infancy as opposed to later childhood. Nor did the sex affect the prognosis, nor, in a small group of 8, an intimate degree of contact with open phthisis.

Another finding of importance in Illing's work is the very close relationship between the miliary disease in the lungs and the presence of enlarged tuberculous glands in the chest. No less than 17 of his 21 cases had obvious radiological evidence of glandular masses, which in 13 involved high paratracheal glands. As these particular glands form the penultimate barrier to a blood-stream infection from the hilar regions, he rightly sounds a note of warning that their enlargement in a tuberculous child is a signal that a miliary catastrophe may not be far off. As we get so little warning, any sign that helps to make a forecast deserves the closest attention; and maybe this is one the significance of which has been commonly overlooked.

C. H.

GENERAL ARTICLES

THE PROGNOSIS OF CHRONIC MILIARY TUBERCULOSIS IN CHILDREN*

By R. B. ILLING
From Grove Park Hospital

The study of hæmatogenous tuberculosis in children is of particular interest, for a large proportion of the deaths in primary tuberculosis are due to a blood-stream spread. Thus Richards, reviewing 445 children suffering from primary tuberculosis or pleural effusions, found that 10 out of 16 deaths were due to miliary tuberculosis or meningitis. On the other hand it is recognised that extensions of the disease by the bloodstream, which are far from fatal, occur not uncommonly in such cases (Ghon); and there is now ample evidence that, even when an extensive dissemination of the disease occurs in this way, the prognosis is not necessarily a hopeless one as was once supposed. However, although many cases of chronic miliary tuberculosis have been published, few writers have commented in more than general terms upon the prognosis or upon the factors which influence it.

By far the most authoritative and comprehensive statement on this subject was made by Hoyle and Vaizey, who reviewed the prognosis of 64 cases, both adults and children. They concluded that their cases fell into three groups. In group one the course was similar to that of acute miliary tuberculosis and death took place in less than six months. In the second group the disease was eventually fatal, but the patients survived for more than six months and remissions lasting up to a year were often present. In both groups death was usually due either to acute tuberculous meningitis or tuberculous toxæmia. The third group contained those cases where the disease became quiescent or arrested. They included in this group only those patients who were observed to be well two years from the last date of symptoms.

In an effort to facilitate the assessment of prognosis in this disease, 21 new cases have been reviewed. They were all children from 1 to 14 years of age, suffering from tuberculosis, who had had a gross hæmatogenous dissemination of the disease into both lungs but had survived for a minimum period of three months from confirmation of the diagnosis by radiographs. Of these cases, 6 were under the writer's care, 5 he was able to observe through the kindness of his colleagues, and the remaining 10 were taken from the records of High Wood Hospital from 1939-1944.

The three-month period mentioned above is a purely arbitrary one. In practice, however, it serves a useful purpose, as survival for as long as this

* Based on a paper read at a meeting of the Tuberculosis Association on January 19, 1945.

separates such patients quite definitely from those with classical acute miliary tuberculosis. It also serves to bring these cases into line with those of other writers who have used a similar method of selection.

Course of the Miliary Lung Lesions

Of the 21 cases, 7 showed no definite change in the X-ray appearances while under observation. These 7 were observed for less than six months, and during that time 6 of them died. The other was taken from hospital against advice after two months' observation.

Twelve of the 21 cases showed radiological retrogression and resolution of the lesions (see Figs. 1 and 2). This was a gradual process which could be followed by serial radiography. In 9 of the 12 it progressed until no definite abnormality could be detected in the final pictures, although the normal lung markings were often more pronounced than usual. The resolution took a variable time to accomplish, but it is impossible to say exactly how long, since radiographs had not been taken at the actual commencement of the disease. The period while under observation varied from eight to twelve months before resolution appeared to be complete.

In one case, following a staphylococcal pneumonia, multiple small cavities developed at the left apex. The miliary lesions, after remaining unchanged in appearance for many months, now appear to be coalescing.

The remaining case healed by the less common method of massive calcification of the lesions (see Fig. 3). The original lesions were larger than is usual and the case is probably one of those described by Armand-Delille as the "lenticular" type. Calcification started after sixteen months of observation and is probably still continuing, three years later.

Course of the Disease

Of the 21 cases, 8 have died. They lived for 3 months, 3 months, 4 months, 5 months, 5 months, 5½ months, 14 months and 7 years and 9 months respectively.

The first 6 of these cases are best described as subacute miliary tuberculosis. They all died in less than six months. The children were toxæmic and developed a hectic temperature. As the disease progressed the temperature and the blood sedimentation rate tended to rise, while there was a corresponding steady deterioration in their general condition from start to finish. Indeed, they differed from acute miliary tuberculosis mainly in a longer period of survival.

The seventh case was exceptional. At her first examination, she was found to have a large, thin-walled cavity in the apex of her left lung besides a diffuse miliary mottling in both lungs. Seven months later a further cavity developed in her right apex and tubercle bacilli were found in her sputum, although the miliary lesions appeared to have partially resolved. Although these lesions continued to resolve, the cavities increased in size and further cavities developed on the left side. Two months after induction of a left artificial pneumothorax she had a left spontaneous pneumothorax; and she died a month later from that with a concurrent broncho-pleural fistula. By

PLATE X.

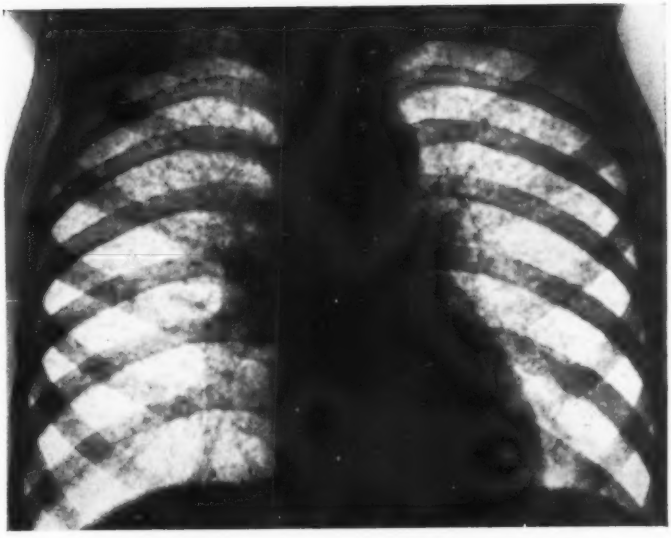

FIG. 1.—FEMALE, AGED 12. CHRONIC MILIARY TUBERCULOSIS.

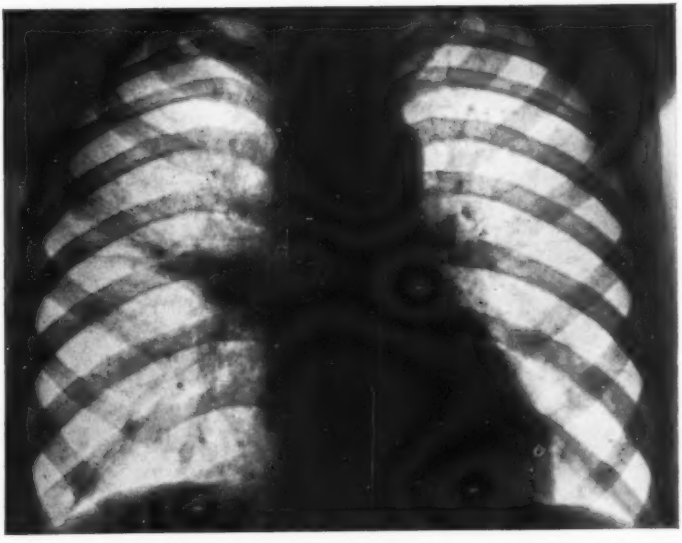

FIG. 2.—SAME CASE AS FIG. 1, SEVEN MONTHS LATER, SHOWING COMPLETE DISAPPEARANCE OF MILIARY LESIONS.

[*To face page* 80.

PLATE XI.

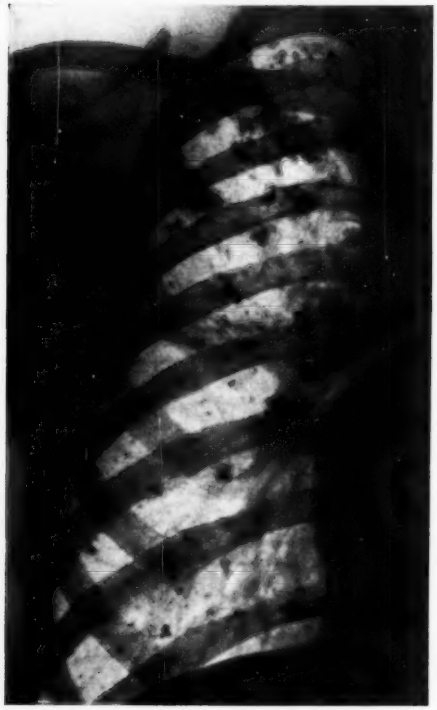

FIG. 3.—FEMALE, AGED 5 YEARS. CHRONIC MILIARY TUBERCULOSIS: DETAIL OF RIGHT LUNG FIELD, SHOWING CALCIFIED MILIARY LESIONS.

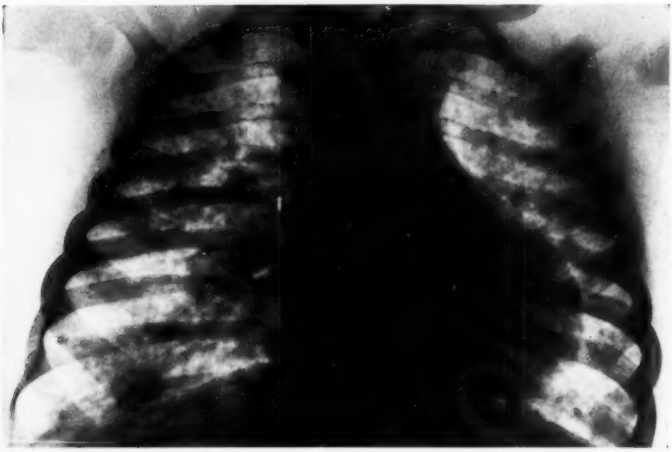

FIG. 4.—MALE, AGED 1 YEAR. CHRONIC MILIARY TUBERCULOSIS: ALIVE AND IMPROVING TEN MONTHS LATER. NOTE ENLARGED RIGHT PARATRACHEAL GLAND.

To face page 81.

that time radiographs showed no signs of any remaining miliary disease, a remarkable happening considering the fatal course.

The eighth case has such particular interest that it deserves more detailed record.

CASE HISTORY.—A girl, aged 12, whose father was known to have pulmonary tuberculosis with a positive sputum, was admitted with a pyrexia of sudden onset which had been present for three months. She had a slight cough, a very small amount of sputum, and looked pale and under-nourished. There were no abnormal physical signs in her chest, but a radiograph showed dense, miliary mottling in all zones of both lung fields with the density greatest towards the apices (Fig. 1). The first sputum examination showed scanty tubercle bacilli. No further bacilli were found in twenty-one subsequent examinations. The Mantoux test was positive (1: 10,000).

Her temperature remained irregular (99-100·6° F.) for the first four months after admission. It then settled for four months, only to rise again when she developed a sternal abscess. Four months later her miliary lesions had cleared completely (Fig. 2) and her temperature had settled once more. During the next fourteen months she had three exacerbations of temperature, each lasting a fortnight, and at the end of that time she had developed spinal caries. She also had albumen in her urine, although no tubercle bacilli could be found.

She was transferred to an orthopædic hospital and treated on a spinal frame for three years. At the end of that time she had bilateral renal tuberculosis. She spent more than eighteen months in two subsequent hospitals, but her general condition showed a steady deterioration. She was finally discharged to her home still deteriorating and she died eleven months later from a terminal tuberculous meningitis (seven years and nine months after the clinical onset of her disease).

Thirteen of the cases are still alive. They have all survived for more than six months: 11 have survived for more than 1 year, 6 for more than 2 years and 4 for more than 4 years—that is to say, for 4 years, 4 years, $5\frac{1}{2}$ years, and $11\frac{1}{2}$ years respectively.

TABLE I.

8 DEATHS AFTER—

3 *mths.*	4 *mths.*	5-6 *mths.*	14 *mths.*	7 *yrs.* 9 *mths.*
2	1	3	1	1

13 SURVIVORS OBSERVED FOR—

6-12 *mths.*	1-2 *yrs.*	2-4 *yrs.*	4-6 *yrs.*	11 *yrs.* +
2	5	2	3	1

AGE GROUPS OF 21 CASES (DEATHS IN PARENTHESIS).

0-2 *yrs.*	2-5 *yrs.*	5-10 *yrs.*	10-15 *yrs.*	*Total.*
4 (1)	4 (1)	6 (1)	7 (5)	21 (8)

AGE GROUPS OF 32 OTHER CASES FROM THE LITERATURE.

0-2 *yrs.*	2-5 *yrs.*	5-10 *yrs.*	10-15 *yrs.*	*Total.*
6 (4)	3 (1)	15 (7)	8 (5)	32 (17)

Factors which Might Affect the Prognosis

In an endeavour to investigate the influence, if any, which age might have on this condition, the 21 cases were divided into four age groups. Thirty-two further cases of this disease in children were taken from the literature and similarly arranged for comparison (see Table I). Although nothing definite can be deduced from such a small number of cases, these two groups show that recovery occurs at any of the ages under consideration and that the mortality appears to be greater rather than less among the older children (10-15 years).

Over the same 53 cases the sex incidence was almost exactly equal (27 boys and 26 girls), and of this total 15 boys and 14 girls were still alive when last observed. There was no evidence, therefore, that the sex of a child has any influence on the prognosis.

It would be reasonable to suppose that any factor which tended to increase the mass of the bloodstream invasion would worsen the prognosis. Close contact with a case of open tuberculosis would be one such hypothetical factor. Of the 21 cases in the present series, 8 had a definite history of close contact with tuberculous relatives. Only 2 of these 8 died, so that here at any rate close contact had no such evil effect upon the outlook.

These eight cases are also interesting in that they were probably due to the human type of bacillus. It has been suggested (Hoyle and Vaizey) that the bovine type of bacillus may be the predominant organism for the chronic miliary tuberculosis occurring in childhood, since Blacklock has shown that the human type of infection is more virulent where generalization of the disease in children is concerned. Unfortunately it is extremely difficult to isolate the organism from cases of miliary disease and no definite evidence one way or the other has yet been presented.

TABLE II.—ASSOCIATED TUBERCULOUS LESIONS

(*Occurred in* 16 *of the* 21 *Cases.*)

Chest.	
Enlarged glands	17 cases
Hilar and paratracheal	11 „
Hilar	4 „
Paratracheal	2 „
Primary focus	2 „
Pleural effusion	5 „
Atelectasis	2 „
Post-primary cavities	2 „
Staphylococcal pneumonia	1 case
Chronic phthisis	1 „
Other Organs.	
Cervical glands	4 cases
Tuberculosis of joints or bone	3 „
Urogenital tuberculosis	1 case
Tuberculides of skin	2 cases
Lupus	1 case
Interstitial keratitis	1 „
Phlyctenular conjunctivitis	3 cases

Associated Tuberculous Lesions

Seventeen cases had enlargement of glands in the chest. In 13 of these the paratracheal glands were involved. In 2 cases a primary focus was also present.

Five cases had pleural effusions and in 1 of these the effusion was bilateral. Four of these 5 died. In 2 of them the effusion was a terminal event, but in 2 others it had probably been present from the commencement of the disease. The case with the bilateral effusion is alive and improving, having been followed for eight months. The prognosis of such cases was considered to be grave by Fernandes, who described 6 cases, 5 of which developed miliary tuberculosis

and died. This sequence is by no means invariable where children are concerned, as shown by the 69 cases of pleural effusion in children analysed by Richards. Four of these had bilateral effusions and all recovered. It is possible, however, that the relatively benign pleural effusions of childhood, even when bilateral, are not usually due to a bloodstream spread, and it is this, when it occurs, which worsens the prognosis.

Two cases had atelectasis. One of these had a right upper lobe segmental collapse which completely re-expanded in four months. The second had a massive atelectasis involving the whole of the left lung, as was confirmed by tomographs, and still present more than three years later. The miliary lesions in the other lung appeared to resolve a year ago.

Two cases developed post-primary cavities. The first had small cavities at the right apex and a positive sputum when first examined. The cavities, therefore, may have preceded her miliary disease. The other one, however, already described, developed cavities while under observation. There is no doubt, therefore, that they did in fact develop from actual areas of miliary disease.

In one case already mentioned, following a staphylococcal pneumonia, multiple small cavities developed at the left apex and the sputum contained staphylococci during the following months. The child is still ill eighteen months after the miliary spread and the lesions appear to be coalescing.

One case, now followed for nearly twelve years, has developed an extremely chronic form of phthisis. Infiltration began to appear seven years after the commencement of her disease and then began to spread slowly. At the same time, three nodules of lupus developed on her face. The lung condition has remained stationary, however, during the past two years. The lupus nodules are still present.

Four cases had enlarged cervical glands. One of these had recurrent enlargement of these glands continuing over four years. She healed her miliary lesions by calcification (Fig. 3) and also deposited calcium in many of the healing glands. Three and a half years after the start of the disease she developed a retro-pharyngeal abscess from breaking down glands and this is still slowly subsiding.

Three cases developed focal tuberculous lesions in other parts of the body. These included: a sternal abscess, spinal caries, bilateral tuberculous kidneys, tuberculous ankle, tuberculous synovitis of wrist and tuberculous synovitis of knee. Two had tuberculides of the skin.

Among other associated conditions there was 1 case of interstitial keratitis and 3 of phlyctenular conjunctivitis.

Discussion

No boundary can be drawn between acute and chronic miliary tuberculosis and every gradation between the most acute and the most chronic type of case can occur in children as well as adults. In general, where chronic miliary tuberculosis is concerned, the intensity and extent of the original bloodstream spread, estimated clinically by considering the child's appearance, temperature chart, pulse chart, weight chart, and the blood sedimentation rate, is the best guide to the immediate prognosis.

With a sudden onset and considerable toxæmia an early death is the likeliest outcome, for it is the toxæmia or a meningitis which kills during the first few months. Improvement, if it occurs, is likely to be temporary. At the other extreme, the " silent " apyrexial type of the disease, often with insignificant symptoms and perhaps discovered only when the child is radiographed as a contact, has a comparatively good prognosis and tends to progress uneventfully to complete cure. Five cases in the present series came into this category.

The intermediate type of case is thus the most difficult to assess. The one described in detail above, who died seven years and nine months after the onset of her disease, provides a good example. In spite of the initial toxæmia, the child improved and the miliary lesions gradually disappeared. But the danger of fresh hæmatogenous disease was shown again and again by a sternal abscess, spinal caries and renal tuberculosis in succession, and finally by a fatal meningitis.

It is impossible to say, with certainty, that the danger of a fresh exacerbation is over in any case of chronic miliary disease. However, Hoyle and Vaizey, after a careful study of 14 cases which had survived for more than six months, but which eventually died having shown at least a temporary improvement, concluded that remissions were only likely to be permanent if they lasted for more than two years. No exception to this conclusion was found among the cases in the present series.

The most likely situation for the focus which gives rise to the initial hæmatogenous disease is a paratracheal gland—the last lymph obstacle between the primary focus and the systemic veins. During the initial hæmatogenous dissemination of organisms a proportion of bacilli probably filter through the network of lung capillaries and, entering the main circulation, pass to any part of the body. Munro suggests that, when focal tuberculous lesions arise later in other organs, they are due to an exacerbation of tubercles formed during this first miliary spread. Fish, however, showed histologically that recurrent spread to the lung does occur, and it is very probable that, at least in some cases, focal tuberculosis elsewhere in the body is caused by a chronic dribbling focus which gives rise to small, recurring, hæmatogenous spreads.

It is interesting to note, therefore, that Fish described a widening of the upper mediastinum in the X-rays of 8 out of his 10 cases, and in 3 of these he found at necropsy that the widening was due to caseating paratracheal glands. Enlarged glands were noted in the chest radiographs in 17 of the 21 cases in the present series, and in 13 of these high paratracheal glands were enlarged (Fig. 4). In one case a paratracheal gland was observed to be greatly enlarged six weeks before the miliary disease occurred. There is, therefore, presumptive evidence that the paratracheal glands may often be the site of the focus responsible for infecting the bloodstream. The point of note that emerges here is that enlarged, high, paratracheal glands in children with primary tuberculosis should probably be regarded as a danger signal and as a sign for careful treatment. Whether miliary disease has already appeared or not the child should be regarded as particularly liable to it until such time as these glands can be seen to have returned to their normal size.

Summary

Twenty-one cases of chronic miliary tuberculosis in children are analysed with regard to the course of their lung lesions, the course of their disease and the incidence among them of other tuberculous conditions.

Assessment of the prognosis for such cases should be divided into two parts: (1) the immediate prognosis, depending on recovery from the initial miliary spread into the lungs, the chances of which are approximately inversely proportional to the degree of toxæmia; (2) The late prognosis, depending upon the development of further tuberculous lesions.

It is suggested that the late prognosis depends mainly on the healing of the bloodstream focus and that this focus is often a caseating, paratracheal gland.

My thanks are due to Dr. J. V. Hurford. Acting Medical Superintendent of High Wood Hospital, for his permission to publish these cases.

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SYPHILIS OF THE LUNG IN BANTUS

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Introduction

SYPHILIS of the lung is considered a great rarity by most writers on the subject. Fishberg stated categorically that it is an extremely rare disease. Howard, in 1927, estimated that there were only 200 cases reported in the literature. Among 2,800 necropsies at the Johns Hopkins Hospital, Osler found only

twelve cases of syphilis of the lung, in eight of which the lesions were congenital. Symmers, in a study of 4,880 necropsies, 314 of which showed lesions due to syphilis, found the lungs involved in twelve and the presence of syphilitic pleural scars in two more. McIntyre, in a review of the subject, states that the literature between 1906 and 1920 yielded sixteen cases in which the diagnosis rested on the results of anti-syphilitic treatment and in twenty-seven cases in which the diagnosis was verified at death.

In our experience syphilis of the lung is far from being a rarity among the Bantus of South Africa. Syphilis is a common disease among them, and in the great majority is either insufficiently treated or not treated at all. Wallace, V.D. Officer for Durban, states that the average number of new cases of syphilis among the Bantus attending for treatment at the Venereal Disease Clinic, Durban, is 500 per month of an estimated Bantu population of 75,000. The number of European new cases is about 30-40 per month of a white population of 107,000. As, however, a great many Europeans are treated by private doctors these figures are not quite a true reflection of the position. In the course of a recent survey in the peri-urban and rural areas near Durban we found an incidence of 19 per cent. positive Wassermann reactions in just over 200 consecutive tests done. Kark and le Riche, in their Bantu Nutritional Survey, found in over 800 blood tests an incidence as high as 46 per cent. in one area. Their average figure for urban areas was 23·6 per cent. and for rural areas 23·3 per cent. These figures are particularly significant when it is remembered that the vast majority of their cases were children between the ages of 7 and 14 years in unselected samples.

The cases of syphilis presenting themselves are very much more serious in the Bantus than in Europeans, mainly because the Bantu does not seek treatment until the disease has become obvious to his employers or fellow workers. Comparatively few Bantus present themselves in the primary stage, whereas most Europeans come in time for the secondary lesions to be avoided by treatment. The disease is more florid and exuberant in the secondary stage in the Bantu and late nerve lesions are comparatively rare.

Pathology

Syphilis of the lungs may be either congenital or acquired. In the latter form, lesions occur both in the secondary and the tertiary stages. In this communication we are concerned chiefly with pulmonary syphilis as it occurs in the tertiary stage.

The middle and lower lobes are said to be most often affected, although cases have been reported where only the upper lobe was the seat of disease. The lesions are usually described as being of two kinds:

(*a*) Gummata.
(*b*) Diffuse Fibrosis.

These lesions are almost certainly tertiary and are obviously not early manifestations of the disease. Stanley (1911) described pulmonary syphilis as consisting of 4 forms:

(1) " Syphilitic Pneumonia "—this he considered the most important form of acquired syphilis of the lung.

(2) An early diffuse sclerosis, the result of (1), in which the lung is not misshapen and is uniformly tough.
(3) Gross scarring of the lung with contraction.
(4) Gumma."

Pearson and de Navasquez in a modern review of the subject conclude that "acute syphilitic pneumonia undoubtedly exists and probably represents the earliest lesion in pulmonary syphilis; that syphilitic pneumonia is primarily interstitial and may lead to (*a*) gummata, (*b*) chronic interstitial fibrosis; and that pulmonary syphilis is always associated with and is secondary to syphilitic aortitis." Boyd also holds the view that pulmonary syphilis is "nearly always associated with and secondary to syphilitic aortitis." In an earlier study, Carrera (1920) had 12 cases of lung syphilis and found syphilitic infection in the heart and aorta in all.

Symptoms and Signs

Syphilis of the lung may simulate any of the more chronic conditions of the lung, particularly pulmonary tuberculosis. The symptoms and signs are those of a chronic inflammatory process, with cough and sputum, bloodstained at times, loss of weight and energy, anorexia and night sweats. The physical signs, none distinctive, depend on the extent and locality of the lesions and the presence of complications.

Radiological Appearances

Pearson and de Navasquez, surveying the radiological findings of various authors, suggested that the more common appearances may be grouped as follows:

(1) Infiltration which may be lobar or patchy in distribution and involving one lobe or part of one or more lobes. There is little or no deviation of the trachea.
(2) Opacities roughly oval in shape, most often involving the lower lobes and often without connection to the hilum. These again may be single and may vary in size from a few millimetres to several centimetres in diameter. Rarefied central areas may indicate cavitation. Strands of increased density may radiate from these areas of infiltration into normal lung tissue.
(3) Generalised accentuation of the bronchial and vascular shadows, often showing a beaded appearance, radiating from the lung roots towards the periphery.
(4) One lobe or the whole of one side of the chest may be opaque with deviation of the mediastinum towards the side of the lesion, indicating fibrosis or pulmonary collapse.

Diagnosis

The diagnosis is made chiefly by the exclusion of other conditions, of which a pneumonia, neoplasm, lung abscess and pulmonary tuberculosis are the most important. As the response to anti-syphilitic therapy is rapid and dramatic

the value of early diagnosis is considerable. Writers generally are agreed that to make the clinical diagnosis of pulmonary syphilis the following points are necessary:

(1) The condition must be a chronic one.
(2) Sputum must be consistently negative for tubercle bacilli on straight smear, concentration and culture, or on guinea-pig inoculation. The sputum must contain no fungi which may cause a similar lesion, or show a similar response to N.A.B.
(3) The serological test for syphilis must be positive.
(4) There must be radiological evidence of a pulmonary lesion.
(5) There must be a rapid clinical and radiological improvement on anti-syphilitic therapy.

Associated signs of syphilis in other organs or a previous history of syphilitic infection may be added as collateral evidence of value on occasion.

Case Reports

CASE NO. 1.—*J. N., Native Male, 48 years, Single, Labourer.*

Present illness began about ten months ago with cough, dry at first and later productive of mucoid sputum. For the last three months his cough has become worse with the production of a fair amount of offensive sputum which during the last month has been bloodstained on occasion. He has lost a good deal of weight. His appetite is poor and he complains of night sweats. *Previous History:* Nil of note. *On Examination:* General condition poor. Temperature 99-102° F. Pulse 100-118. Respiration 20-24. Pallor of visible mucous membranes. Clubbing of fingers present. No adenopathy. No evidence of previous skin lesions. *Respiratory System:* Left lung—dullness, bronchial breathing and crepitations in lower half. *Cardio-Vascular System* and *Central Nervous System:* Nil abnormal found. *Alimentary System:* Nil abnormal found. *Sputum:* Negative for tubercle bacilli. *X-ray of Chest* (Fig. 1): Showed a massive lesion in the left lower lung field with cavitation in its upper part. *Blood W.R.:* Strongly positive.

Course.—The radiological appearances suggested a lung abscess. He was given a full course of sulphonamides and short wave diathermy to his chest, with no clinical nor radiological improvement. He was then given N.A.B. and Bismuth with a rapid and dramatic improvement. Within two months of commencing anti-syphilitic therapy his chest was practically normal (Fig. 2). He felt so well, with complete loss of symptoms, that he insisted on going home. He was discharged from hospital and referred to the V.D. Clinic for further treatment.

CASE NO. 2.—*R.N., Indian Male, 49 years, Labourer.*

About six months prior to admission to a neighbouring hospital he began to complain of cough and sputum and pain in the left side of the chest. These symptoms have steadily become worse. His sputum has been bloodstained on occasion. He has lost weight. His appetite is poor and he complains of lassitude and disinclination for work.

On Admission: His general condition was poor. He looked ill. Temperature 97-99° F. Pulse 88-96. Respiration 20-22. Weight 126 lbs.

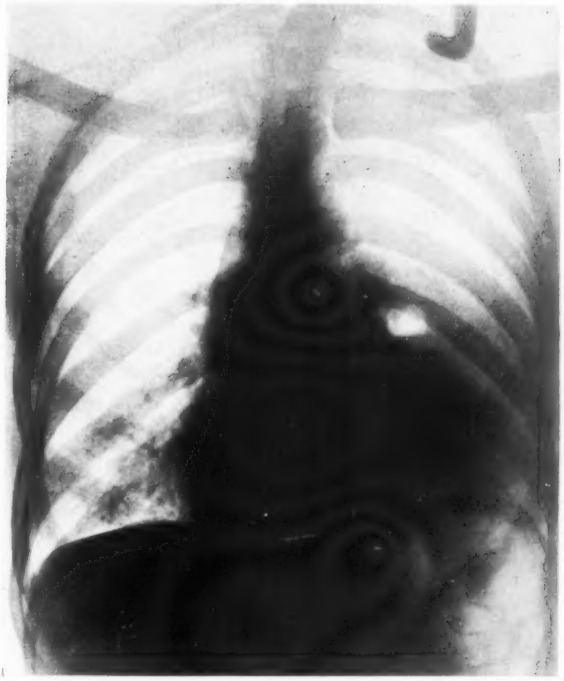

FIG. 1.—(CASE 1) MASSIVE LESION WITH CAVITATION IN THE LEFT LUNG.

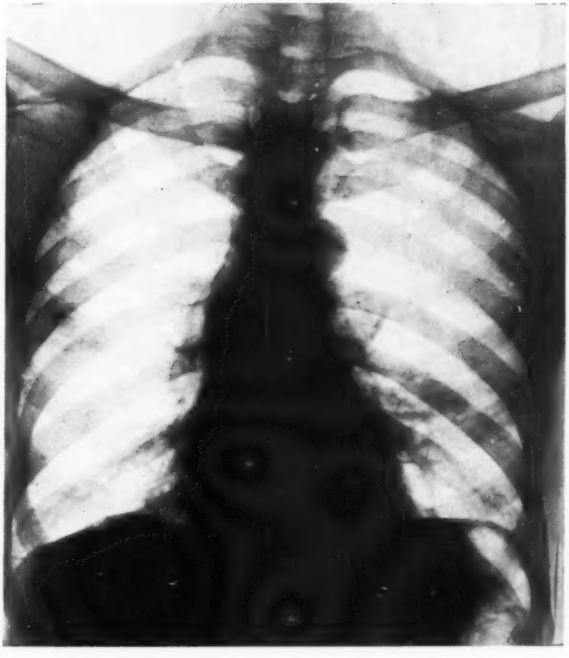

FIG. 2.—SAME CASE AFTER TWO AND A HALF MONTHS TREATMENT.

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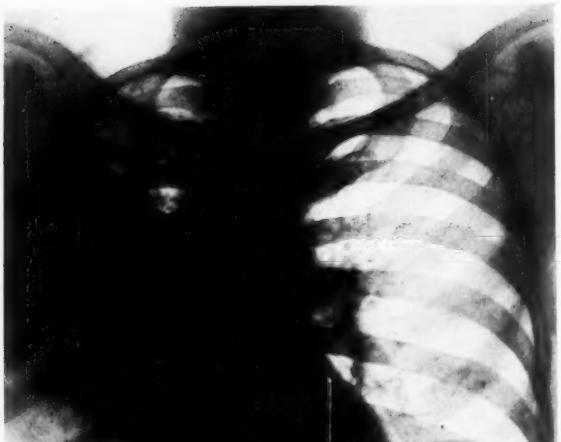

FIG. 3.—(CASE 3) MASSIVE CONSOLIDATION IN RIGHT LUNG.

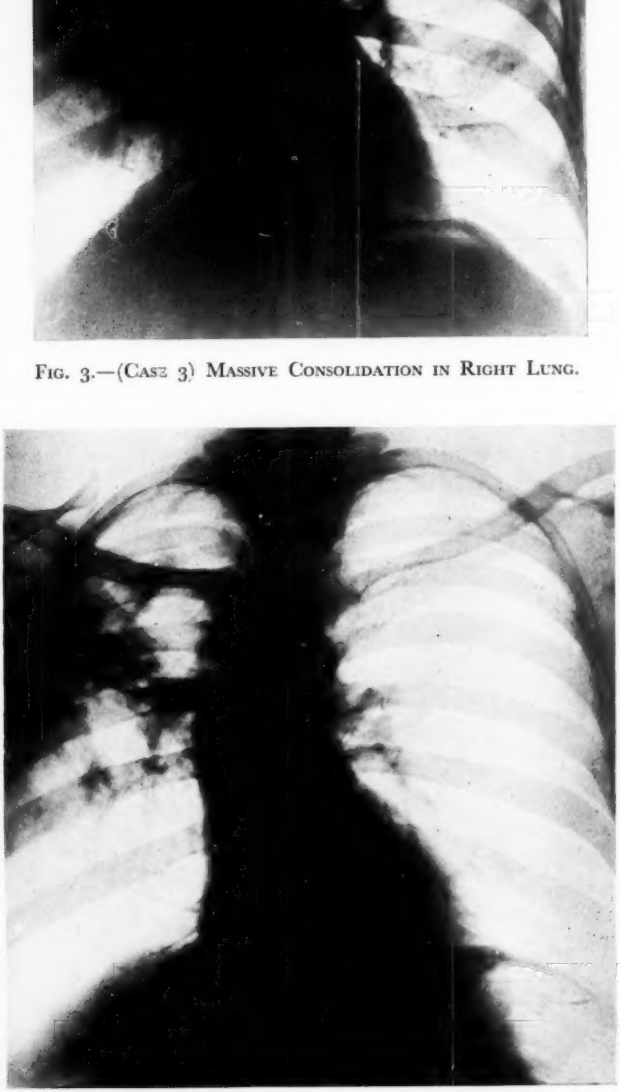

FIG. 4.—SAME CASE TWO AND A HALF MONTHS LATER AFTER TREATMENT.

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Respiratory System : Slightly dyspnœic at rest. Slight clubbing of fingers. Scattered rhonchi in both lungs. Crepitations at left base. *Cardio-Vascular System :* Nil abnormal found. Blood Pressure 118/70. *Abdomen :* Spleen palpable. Liver not felt. *Central Nervous System :* Nil abnormal found. *Blood Examination :* Hb. 80 per cent. C.I. 0·94. R.B.C. 4,250,000/cu.mm. W.B.C. 8,600/cu.mm. Diff. Polys. 78 per cent. Lymph. 17 per cent. Eosin. 2 per cent. Monos. 3 per cent. No malarial parasites seen. *Blood W.R. :* Strongly positive. *Sputum :* Repeatedly negative for tubercle bacilli. *Urine :* Normal. *X-ray of Chest :* Shows opacity with central cavitation in the midzone of the left lung extending from the root area.

Course.—He was given N.A.B. and Bismuth, but after only 8 weeks of treatment he insisted on going home as all his symptoms had disappeared and he felt fit. A further X-ray showed an almost complete disappearance of the lesion. A diagnosis of breaking down gumma of the lung was made in view of the rapid and remarkable response to anti-syphilitic therapy. Unfortunately he could not be followed up as he left for the country.

Comment.

The final diagnosis in this case (as in the previous one) was based on the therapeutic result from N.A.B. and Bismuth. The radiological appearances in both suggested a chronic lung abscess. The question naturally arises: were these examples of chronic lung abscess in syphilitic subjects rather than breaking down gummata with central necrosis? Although we cannot altogether exclude the former we are inclined to the latter view. We are not the only ones to have tried N.A.B. in lung abscesses in non-syphilitic subjects with very little, if any, radiological or clinical improvement. The response to anti-syphilitic therapy in these last two cases was both rapid and dramatic—too rapid, we feel, for their lesions to have had a non-syphilitic basis.

CASE NO. 3.—*D. M., Native Male, 48 years, Labourer.*

This man was admitted to a neighbouring hospital with a history of cough and pain in the right chest for six weeks.

He says he has been coughing for " many months," but the last six weeks his cough has been worse and he developed a pain in the right side of his chest, chiefly in front. His sputum is mucoid but has been bloodstained on occasion. He has been losing weight and appetite; there has been a progressive loss of energy. He also complains of night sweats.

On Examination : Temperature, pulse and respiration normal. General condition fair—looks thin. Tongue furred. Mucous membranes fairly well coloured. No rash. No adenopathy. *Respiratory System :* Dullness over right upper lobe. Impaired air entry with scattered crepitations on the right side. *Cardio-Vascular System* and *Alimentary System :* Nil abnormal found. *Blood W.R. :* Strongly positive. *Sputum :* Negative for tubercle bacilli. *X-ray of Chest* (Fig. 3) : Shows a very massive lesion on the right side occupying practically the whole of the upper two-thirds of the right lung field.

Course.—A further X-ray of the chest after two-and-a-half months N.A.B. and Bismuth shows marked clearing (Fig. 4). The patient absconded, and all trace of him was lost before further treatment could be given.

CASE NO. 4.—*J. X., Native Male, 49 years, Houseboy.*

A married man with two children, whose wife had also had one miscarriage. Seen at a neighbouring hospital complaining of a cough. He was unable to

give the duration of his illness, but said it was a " long time." His illness first began with a certain amount of lassitude and loss of appetite, followed some little time after with a dry cough which later became productive of mucoid sputum. Apart from the cough he said he felt quite well.

On Examination : General condition fair. Good colour of visible mucous membranes. Old traumatic cataract right eye. Clubbing of fingers. No adenopathy. No evidence of present or past skin lesion. *Respiratory System :* Dullness with impaired air entry at right upper lobe. *Cardio-Vascular System :* Soft systolic murmur at apex, otherwise nil abnormal found. Blood pressure 148/98. *Central Nervous System :* Reflexes intact, no sensory loss. Testicular sensation present. *Sputum :* Tubercle bacilli not found on straight smear and on concentration. No fungi seen. *Blood W.R. :* Strongly positive. *X-ray of Chest* (Fig. 5) : Shows a fairly massive lesion in the right upper lobe, like a consolidation. The aorta is widened, the knuckle lost, and the left border of the heart prominent as from slight enlargement.

Course.—A tentative diagnosis of pulmonary syphilis was made and he was put on to anti-syphilitic therapy. Potassium Iodide grs. xxx. t.d.s. was given for three weeks, after which Mapharside was administered. After receiving eight injections (total 0·34 gms.) he completely lost his cough and had gained 8 lbs. in weight. His appetite and a sense of well-being returned fairly rapidly and he insisted on being discharged from hospital. A radiograph of his chest on discharge showed almost complete clearing of the lesion in the right lung (Fig. 6). The dilatation of the aorta is much better seen. He was advised to attend the V.D. Clinic for further treatment.

Discussion

Although so much has been written about pulmonary syphilis most critics retain an air of scepticism as to the validity of the criteria upon which it is often diagnosed. Perhaps their reluctance to admit the clinical entity of pulmonary syphilis is due to the fact that the disease is well treated in its early stages in most countries today, and so the florid forms of syphilis are but rarely encountered. We happen to live among primitives who still show those florid manifestations seen by our grandfathers and great grandfathers in Europe; and we undoubtedly see a pulmonary disease which by a process of exclusion can be regarded as pulmonary syphilis. There is no pathognomonic sign nor characteristic syndrome. Cough, sputum, loss of weight, night sweats and pleural pain are all common and in a chronic way. Yet no tubercle bacilli or pathogenic fungi can be found, serological tests for syphilis are positive, and radiographs show unmistakable evidence of severe parenchymal disease in the lung that rapidly improves concurrently with anti-syphilitic treatment. There is, of course, the fallacy of spontaneous remission of some form of virus or suppurative pneumonia to consider when the disease can only be regarded as recent. But this becomes less likely with a continuity of symptoms going back for many months as in the cases cited here. Unfortunately the opportunity for confirmation pathologically has not yet come our way, for all these cases recovered rapidly on arsenicals.

It seems quite likely that pulmonary syphilis among the Bantus is not rare but is usually overlooked. Further light on the matter would quickly be forthcoming if a routine radiograph of the chest were taken in all those with syphilis who develop any pulmonary symptoms.

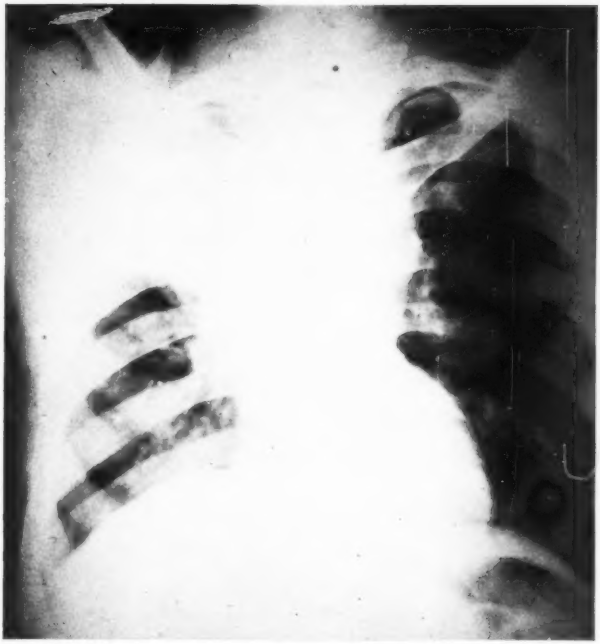

FIG. 5.—(CASE 4) CONSOLIDATION OF RIGHT UPPER LOBE AND EVIDENCE OF AORTITIS.

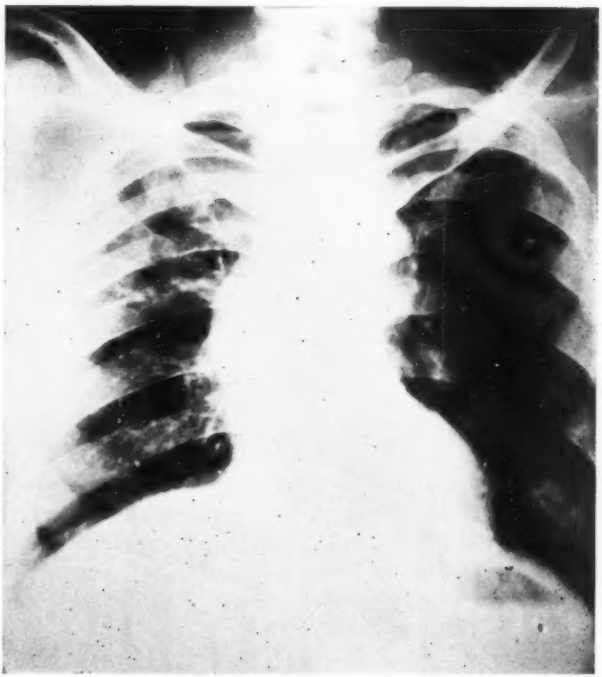

FIG. 6.—SAME CASE AFTER TWO AND A HALF MONTHS TREATMENT.

[*To face page 90.*

Summary

Four native patients, believed to have syphilis of the lung, are described. They all improved rapidly on anti-syphilitic treatment although without exception they had been ill for many months before. The condition is probably less uncommon among the Bantus than among white races, for they still acquire florid forms of syphilis.

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THE ÆTIOLOGY OF SILICOSIS

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From the Camborne-Redruth Hospital

The extensive study of silicosis which has been made in Europe, America and South Africa during the past two decades has added greatly to our knowledge of the disease and may indeed prove to have gone some way toward finding a solution of the problem which it presents. As the literature on this subject is drawn from the fields of research in mineralogy, chemistry, physics and other applied sciences, a review of recent studies on the ætiology of silicosis and their correlation with work of earlier date may be of interest to physicians who deal with pulmonary disease.

Early History and Natural History

There is good reason to believe that silicosis may be the earliest of all occupational diseases. In England neolithic man sank shafts into the earth (*e.g.*, Grimes Grave in Norfolk, *c.* 5,000 B.C.) from the bottom of which " levels " were excavated for getting the best workable flints for implement making. The flints were carried to the foot of the shaft and were there chipped to the required shape and sharpness. The resulting conditions, prolonged exposure to a highly siliceous dust in an ill-ventilated atmosphere, would be identical

with those which have produced silicosis among " flint-knappers " in the twentieth century.

The condition may be said to have been described first by Hippocrates, who noted the occurrence of breathlessness in certain metal grinders, who " declined rapidly."

Silicosis was also known to physicians in Renaissance times (*e.g.*, Paracelsus), being included by them under the generic title of " phthisis," which signified any chronic disease of the lungs accompanied by expectoration and emaciation. But they realised that it had a relation to occupation.

The disease came into renewed prominence in South Africa in the gold-mining days of the 1890's, its emergence synchronising with the increased use of blasting and the development of the mechanical rock drill, a significant conjunction to which reference will be made later.

The first modern systematic work was that done by a British committee under Dr. J. S. Haldane in 1902, and it was formed to investigate this disease among Cornish tin miners. This committee first stated that stone dust was the cause of silicosis and drew attention to its frequent relation to pulmonary tuberculosis.

Collis[1] in 1915 first observed that not all dusts had the effect of causing silicosis. He felt that the sharpness of the particles was not the cause of the disease, but could not offer an alternative explanation.

Gye,[2] Purdy, and Kettle[3] in 1922 proved that colloidal silica is toxic when injected into rabbits, and suggested that crystalline silica dissolves in the tissues and produces this toxic colloid form. This work forms the basis of the present-day conception of the pathogenesis of silicosis.

Silicosis may be defined as a disease due to breathing air containing silica, characterised anatomically by a generalised pulmonary fibrosis and the development of miliary fibrous nodulation in the lungs.

Silica is one of the most widely distributed constituents of the earth's crust, of which it forms about 30 per cent. Sixty per cent. of all rocks are siliceous, the commonest examples being quartz and sandstone. The only common non-siliceous rock in Britain is limestone.

Of some importance is its prevalence in marine life, as the main skeletal constituent of the diatomic marine plants. The debris of these diatoms sedimented to the sea bed and subsequently uncovered by the recession of the sea in prehistory results in the existence of deposits of diatomaceous earth, or Kieselguhr, an abrasive substance much used in industry and containing 96 per cent. uncombined silica.

This widespread occurrence of silica and its constant use in new commercial processes makes close enquiry necessary before assuming that a person has not been exposed to silica dust. A classical example is that quoted by Middleton,[4] of a man engaged all his working life in making glass-cutting wheels (plumber's diamond), who died of silicosis with coalescent nodulation and tuberculosis after fifteen years' work. He sharpened the glass cutters on a fine grindstone, whose constitution was found to be almost pure silica, which gave off a dust almost imperceptible, but nevertheless containing 10 million particles per cu. ft.

All normal lungs contain dust, a fact well exemplified by the dark lungs of

town dwellers, and this inevitably includes a certain amount of silica, up to 0·1 per cent. dry weight. Amounts in excess of this should be regarded as pathological.

There are other conditions than silicosis which result from dust inhalation, and may show some diffuse fibrosis of foreign-body type in the lung framework —*e.g.*, pure anthracosis due to coal dust—but they do not cause nodulation, only silica will do this. Leroy Gardner,[5] who has made a great contribution of detailed work to this subject, does not consider these conditions pathological, and has suggested the name " benign non-specific pneumoconiosis " for them. He states that when a coal or iron-ore miner develops silicosis, it is the result of silica present as an impurity in the material mined, and the condition should be called anthraco-silicosis or sidero-silicosis.

Silicosis is characterised by its faculty of progressing for long after the subject's withdrawal from exposure to dust.

A condition midway between the benign pneumoconioses and silicosis is asbestosis, due to the inhalation of asbestos particles. Asbestos is got by mining, and is a fibrous compound silicate of magnesium and other bases. It produces a diffuse fibrosis without nodulation, and without progression when exposure ceases; but it gives rise to definite disability.

Bagassosis,[6] an occupational respiratory disease of workers handling dry sugar-cane pulp (bagasse), and consisting of a pneumonic phase and a subsequent disabling pulmonary fibrosis, illustrates the manner in which a pneumoconiosis due to dust of vegetable origin may still come within the scope of Gardner's classification, for the dust of bagasse has been found to contain 7 per cent. silica.

Normal and Morbid Anatomy

In quiet respiration the normal person inhales about 12,000 litres of air per twenty-four hours. It is clear that without an adequate protective mechanism the lungs would speedily be clogged with dust in a normal atmosphere.

Two mechanisms are involved in keeping the lungs clean, one to prevent dust entering them, and the other to remove dust that does enter.

The first mechanism depends on the wet and sticky mucous membrane, peristalsis in the smaller bronchial tubes and the ciliary sweep which propels dust back from the bronchial tree to the pharynx, and the act of coughing (the efficiency of which is dependent on a sound condition of the thoracic wall). A further factor may be, as suggested by Lehmann,[5] that the shape of the nasal air passages conduces to trapping dust in the naso-pharynx. It is known that if dust be blown through a tube which is constricted at its middle, the majority of the particles come to rest just distal to the constriction. He suggests that the nasal air passages are similarly constricted in the region of the turbinate bones.

It may therefore be stated that conditions such as chronic sinusitis with post-nasal drip, or repeated catarrhal bronchial infections, which temporarily or permanently destroy cilia, will predispose to silicosis. It has been shown, too, that the inhalation of certain acid gases liberated by the firing of explosives

paralyses the bronchial cilia. Mouth-breathing must also be considered a factor, and is almost inevitable in a man working hard.

Dust inhaled into the lung is conditioned by two factors: primary distribution, which has been little investigated, and would seem to be a function of the inspiratory air currents; and secondary deposition, on which a great deal of work has been done.

This secondary deposition is the function of the second mechanism, that for removing dust which has entered the bronchioles and the alveoli and is no longer in contact with ciliated epithelium. It depends on the pulmonary phagocytes and the lymph vessels.

These phagocytes originate in the wall of the alveolus and are probably wandering connective tissue cells (histiocytes), though they may be of epithelial origin. The number of them found in the alveolus is proportional to the number of dust particles present, and having ingested the particles they move to the lymph vessel which forms a collar to the neck of the air-cell, and enter the vessel by traversing the intercellular spaces. From this point the removal of the particles is the work of the lymphatic system.

We may recall that the lung has two sets of lymph vessels, one running over its surface in the substance of the visceral pleura, and the second a deep system located in the connective tissue sheaths of the bloodvessels and bronchi. The two systems communicate by short, thick connecting vessels which run in the septa between the subpleural lobules, so that lymph can flow from the deep to the superficial system if the former is obstructed.

Wherever lymph trunks of any size connect there are small aggregations of lymphoid tissue, and these lymphoid aggregations are larger the nearer they lie to the hilum.

Both lymph systems discharge most of their lymph into the tracheo-bronchial lymph glands, but some efferents also pass through the crura of the diaphragm and enter the glands about the cardiac end of the stomach.

It is also apparent, therefore, that any disease process (*e.g.*, pneumonia) which affects the alveolar wall, the lymph vessels, or the lymphoid aggregations will, by producing scarring in these delicate structures, impair their function of carrying foreign matter to the glands, and will thus predispose to silicosis. This is a significant point in considering the question why, under old working conditions, some men (25 per cent.) developed silicosis, while others similarly exposed to dust did not.

It is in the phagocytes that the specific effect of particles of silica is first seen. Free silica quickly injures them, and does so in direct proportion to the number of particles present in the phagocyte and more especially to the smallness of the particles, the small ones having a much greater surface in relation to their mass.

The phagocyte containing ingested silica shows a sequence of events closely resembling that in a tubercle; the cell enlarges, the cytoplasm increases, lipoid droplets are distinguishable in the cytoplasm, and later the nucleus may divide repeatedly, resulting in the formation of a typical Langhans giant cell. Some of the phagocytes, in course of passing through these stages, appear to become "mummified," their nuclear structure ceases to take stains, but the cell envelope may not disintegrate for a long time and the silica particles remain within it.

Asbestos fibres are too long (10 microns and often very much longer) to be ingested by a phagocyte, and a part of the fibre often protrudes out of the cell, or several phagocytes may be seen attached to one fibre. The phagocytes are unable to bear the fibres away into the lymph spaces owing to their largeness, and they remain applied to the wall of the alveolus and terminal bronchiole and fibrosis occurs at these sites. But it is not the typical collagenous fibrosis of silicosis, and nodulation does not occur.

The essential feature of the silicotic process is an *excessive* fibrosis in the intrapulmonary lymphoid aggregations (compared with the insignificant fibrosis of the benign pneumoconioses), together with a fibrosis of the perilymphatic connective tissue, and a hyaline modification of the fibrous tissue, due to the specific effect of silica. The nodules so formed impede the flow of lymph.

When a certain degree of this fibrosis has occurred, the phagocytes appear to become less able to remove particles from the alveoli into the lymphatics (perhaps owing to increased pressure in the lymphatics), and they begin to accumulate in groups on the alveolar walls. Here again a proliferation of connective tissue cells occurs and parenchymatous fibrosis develops.

When nodulation has developed, the nodules increase in size by the deposition of fresh layers of fibrous tissue at their periphery, in response it is believed to the toxic effect of the silica particles within them and of others carried to them by neighbouring lymph vessels and arrested at their margins. Adjacent nodules fuse together, and there results the familiar massive conglomerate silicotic lesion often seen post mortem in miners. The nodules are tough, even hard, and contain gritty-feeling foci, and there is a zone of black surrounding them, or indeed the whole nodule is often black, owing to the presence of carbon particles trapped among the fibrous tissue. The lungs of the silicotic who has survived longest tend to show the least amount of particulate silica post mortem, and *vice versa*.

Associated pathological features are the development of emphysema at the lung margin, especially in the costophrenic area, perhaps owing to traction of the fibrous tissue in the deeper parts, and shrinking and hardening of the tracheo-bronchial glands.

This pathology accounts for the fact that while pulmonary tuberculosis is among those respiratory conditions which predispose to silicosis, silicosis renders a man more vulnerable to tuberculosis. His lungs are poorly aerated, fibrosis results in under-vascularisation, and in the formation of little bronchiolectases and areas of ill-drained alveoli which pocket stagnant secretion and facilitate any infective process. Even in modern hygienic conditions, 75 per cent. of silicotics die of pulmonary tuberculosis.

Occupational Considerations

The occupational factors concerned in the ætiology of silicosis have been admirably surveyed by Middleton,[4,7] and the diversity of occupations carrying a silicosis hazard is impressive.

In Great Britain there are large areas where silicosis is still common—*e.g.*, the potteries, the Derbyshire lead mines, the iron-ore mines of Cumberland, and the recently investigated South Wales anthracite and coal mines.

Among the main silicosis-producing processes are:

(*a*) *Sand-blasting*, in which a stream of quartz sand or crushed calcined flint is projected with tremendous power on to metal articles in order to smooth their surface. This was, until the introduction of stringent protective measures, one of the most fertile causes of silicosis.

(*b*) *Metal-grinding*, as in the cutlery trade. In England grindstones of sandstone were almost invariably used for this purpose.

(*c*) *The pottery and earthenware trade.*—In the making of porcelain and earthenware the main source of dust is the flint sand in which the soft clay articles are bedded for baking after being moulded.

It has been shown, too, that high concentrations of siliceous dust may be found in the moulding rooms. The clay used in moulding is plastic and therefore safe, but fragments of it fall on the floor and on the workers' clothes and after drying form dust.

It is stated that in the last century it was rare to find a working potter who had not become disabled by " potters' rot " by the age of forty.

(*d*) *Abrasive soap and polish making.*—In this industry the abrasive material is usually fine quartz sand, diatomite, " Neuberg chalk," or other forms of naturally occurring silica; these are mixed with powdered soap, sodium carbonate, etc. Middleton has detailed a series of cases of an acute form of silicosis occurring among workers in an abrasive soap factory which operated for a period of eight years; 13 of the 59 persons employed are reported to have died within the next eight years, the cause of death being silicosis or tuberculosis. Middleton stresses the important point that such industries as this are likely to be started on a small scale, by employers who have no knowledge of the risk they carry.

(*e*) *Refractories Industry.*—Silica bricks, moulder's composition, etc., for use in the making of steel castings, are made from ganister, a sandstone containing over 90 per cent. silica.

(*f*) *Quarrying.*—The quarrying of sandstone, granite, and slate is recognised as an occupation frequently causing silicosis..

(*g*) *Metal Mining.*—Tin, lead, iron-ore, and gold are the metals chiefly concerned, the first three named causing silicosis in Great Britain. Gold mining is of considerable interest in that large numbers of Cornish tin miners and Cumberland hæmatite miners have in the past supplied skilled labour for the South African and other gold-fields, and many of these men have returned home with silicosis.

(*h*) *Coal Mining.*—It was believed that the only silicosis risk in this industry was to those men engaged in " hard headings," " roof-ripping," and similar work involving the breaking out of rock, and to colliers in the anthracite mines.

The recent work of Hart,[8] Aslett, and Belt has shown, however, that in the South Wales coal field there is a considerable silicosis risk in the soft-coal mines and that this is greater in the steam-coal mines and greatest in the anthracite mines. They have shown that the fibrosis in this condition (arising from the inhalation of siliceous dust mixed with coal) is of a more diffuse type and with less nodulation than that of metal miners.

In addition to the above industries it is well to remember that men who have worked in any form of boring or tunnelling are liable to develop silicosis.

The Dust

When we consider the physical and chemical aspects of dust inhalation we cannot but feel surprised that silica should be the causative agent of disease. As King[9] has remarked, in view of its apparent insolubility and its persistent inertness in inorganic chemistry, one would expect it to be about the last thing in the world to have a toxic effect.

It is not surprising, therefore, that the earliest workers took the view that the disease was a fibrosis due to a sustained microscopic trauma inflicted by sharp particles in the constantly moving lung.

This theory was upset by the observations (*a*) that other sharp-particle dusts—*e.g.*, metal dusts—do not cause nodular fibrosis, and (*b*) that when the silicotic process had existed for many years particles were frequently not discoverable in the lung post mortem.

Next it was proved that silica, until then considered insoluble, was in fact soluble if its particles were reduced to a sufficiently small size, and that this solubility was enhanced if the reaction of the solvent was weakly alkaline. Controversy still exists as to whether this silica is in true molecular solution or in colloidal suspension, and it seems possible that silica may be brought into both of these states. In either event it would seem that this work provides an adequate basis for the " solubility " theory of the pathology of silicosis, and we will accordingly refer to " silica in solution " as including either form.

Much thought has been given by many workers to the collection and analysis of industrial dusts; indeed, it may not be immediately apparent how important the first of these considerations may be.

Briscoe[10] states that dust samples should be collected exactly as breathed by the worker—*i.e.*, from the air, at breathing level, not moistened by any form of wet-trap, and not subjected to gross changes of temperature. Dust concentrations should be expressed as the number of particles per cubic unit, and not by weight, because the largest particles weigh most and matter least, being too large to enter the pulmonary alveoli and less soluble than the small particle.

It may be said with some truth that danger lies rather in the dust which cannot be seen than in that which can.

The great majority of particles found in the alveoli measure less than 5 microns and very rarely do they exceed 10 microns, save in the case of asbestos fibres, which may measure 100 microns and more.

The silica particles found in silicotic lungs are of the order of 1 to 3 microns.

Rock of mixed constitution will not, when worked, necessarily give a dust of corresponding constitution—for this is influenced by the varying hardness and brittleness of the constituents. Similarly dust found on rafters or floors will not necessarily correspond to an air-floated sample, as it will contain a relatively higher proportion of heavy particles.

If a dust is of uniform composition the factors of particle size, specific gravity, capacity to absorb water, and agglutinability, are largely decisive in determining its settling-time.

It is found that particles smaller than 1 micron settle in still air at 1 to 3 ft. per hour. Above 5 microns the settling-rate is more definitely influenced by

the specific gravity, to which it is directly proportional. Above 10 microns (excepting asbestos fibres) settling is so rapid that such particles have little importance in dust formation.

Dust particles agglutinate in a similar manner to that of bacteria, etc., in biology, and the process is important inasmuch as when a number of particles have clumped together they will settle more rapidly. The agglutination process depends on electrostatic charges and hygroscopic properties in the particle, and it can occur between particles of dissimilar nature. It will be appreciated that an admixture of suitably charged particles with the silica particles is capable of producing increased agglutination and more rapid settling.

It is to be noted that microscopic methods are not at present capable of distinguishing the nature of crystals measuring less than 2 microns; this must be established by chemical analysis or X-ray diffraction methods.

Experimental Researches

Work on animals has largely followed two lines, injection experiments and inhalation experiments.

One of the earliest discoveries in this field was that of Kettle, who noted that the then newly available solution of silica was toxic when injected into rabbits, causing almost immediate death if a sufficient dose was injected intravenously. He suggested that in view of this fact it might be that crystalline silica in the pulmonary alveoli was dissolved in the body fluids (which have the necessary weak alkaline reaction) and thus became toxic.

Next Gardner,[11] injecting suspensions of small particles of pure silica in normal saline, showed that they invariably produced a progressive fibrosis of true silicotic (collagenous) type. He gave repeated intravenous injections to his animals (to exclude the fibrosing effect of local trauma), and was able to produce this reaction not only in lung, but in any organ which contained connective tissue cells.

Working with suspensions of different forms of uncombined silica, Gardner was able to produce figures indicating the comparative fibrosing effect which they produced; thus quartz crystals gave a fibrosis represented by 5+, calcined flint crystals 5+, colloidal silica in the "dispersed" form 8+, and colloidal silica in the "gel" form only ±.

He confirmed Kettle's observation that colloidal silica in the dispersed form quickly causes death when injected intravenously, but found that in the "gel" form (produced, *e.g.*, by changing the reaction of the suspension) it was innocuous; even though the particles could be seen contained within phagocytes, no fibrosis had ensued after the passage of two years.

Repeating these experiments with suspensions of the natural minerals, Gardner found that they were much more reactive than his suspensions of pure silica. Further observations which he made were that both the rate and extent of fibrosis are in inverse proportion to the size of the particle, those between 0·5 and 1 micron being most reactive, and that particles above 10 microns cause a standard degree of fibrosis with no tendency to progression. Particles smaller than 0·002 micron were not retained in the tissues, caused no reaction, and were excreted in the urine.

He noted that asbestos particles when injected produced no fibrosis, and in view of the facts that the pulmonary fibrosis of asbestosis is not of hyaline type, and does not progress after exposure to the dust ceases, he inclines to think that this fibrosis is perhaps of mechanical type, as silicosis was at first thought to be.

Jones[12] in 1933 examined the residue of twenty-nine silicotic lungs, and stated that silica was not the cause of silicosis, but that it was due to particles of sericite, a compound silicate of potassium and aluminium, widely distributed in siliceous deposits in nature.

He based this belief on the findings (*a*) that the bulk of the residue from his series of lungs was sericite, and that sericite was abundantly present in the dust which had been inhaled by the workers, (*b*) that the number of quartz particles that he found was small by comparison with the sericite, and (*c*) that he had observed that the working of rocks containing a large proportion of fibrous silicates such as sericite caused a high incidence of silicosis.

Haldane and Kettle, discussing this paper, pointed out the weakness of the assumption that because mineral particles were found in the fibrosed lung post mortem they were the cause of the fibrosis. They suggested that the persistence of the sericite might be an index of its inactivity and that the more reactive silica particles might have been dissolved during the years that the condition lasted.

In this connection it is noteworthy that white china clay contains much sericite, with very little uncombined silica, and that although no precautions are taken in working it silicosis is very rare among china clay workers.

Robertson[13] and co-workers investigating Jones' theory have concluded that silica rather than a silicate is the material concerned in causing silicosis.

Gardner, too, in his injection experiments, found sericite one of the most inert substances tested, causing no fibrosis.

Another form of combined silica, carborundum (silicon carbide), though its particles are exceedingly hard and sharp, produced no fibrosis when used either in injection or inhalation experiments, and it is noteworthy that the substitution of carborundum grindstones for sandstone ones has largely abolished silicosis among Sheffield cutlery workers.

Silica is excreted in the urine, but owing to the minute quantities present in blood and urine, and a number of variable factors difficult to control (*e.g.*, silica in diet) no convincing results have at present been forthcoming from attempts to compare blood silica content and silica excretion between silicotics and normal controls. Carefully controlled investigations of silica balance have not yet been made.

King has suggested, however, that the soluble silica is fixed in the collagenous fibrous tissue of the nodules, for he found no excess of silica in the blood of silicotics; and if the soluble silica is not fixed in the lung, but is carried to the kidney and excreted, it cannot be in the toxic form, for there is no renal fibrosis.

Gardner observes that despite the strong evidence supporting the " solubility " theory, he has found that quartz and diatomite have an equal fibrosing effect in animal experiments (5+), but their respective solubilities are 5·2 and 35 parts per million. Also different samples of naturally occurring quartz vary

widely in their solubility, but it has never been shown that there is a corresponding variation in their fibrosing effect.

It has been known for long that the solubility of silica shows variations when the silica is mixed with other substances, some of which have been called "solubility depressors." This variation is also seen in the pathogenicity and was first referred to when Haldane[14] noted the apparent modifying effect of coal dust when inhaled with silica. Certain calcium salts also have this modifying effect; thus cement workers, inhaling a mixture of free silica with lime, very rarely develop silicosis, though no dust-control precautions are taken.

It is noteworthy that experiments depending on the solubility of silica and made *in vitro* (either with aqueous solvents or body fluids), or on tissue other than lung-tissue in animals, are not necessarily comparable to inhalation experiments, in which the hydrogen ion concentration in the lung is constantly varying.

For this reason a number of elaborate dust-inhalation experiments have been performed, perhaps the most interesting being those of Denny, Robson and Irwin, working in Toronto.

The late Sir William Bragg[15] and other workers, using X-ray diffraction methods, have shown that the atoms of crystalline structures are arranged in a definite three-dimensional pattern—*e.g.*, the carbon atoms in diamond are arranged in tetrahedra, each tetrahedron having another carbon atom at its centre, and every atom forming the centre of another tetrahedron. The linkages between the atoms are thought to be of an electrostatic nature. These workers have shown that when such a substance is brought into its liquid or gaseous form the atoms are more free, but on reverting to the solid state they seek always to return to their three-dimensional lattice form, in which their valencies are fully satisfied.

Heffernan[16] suggested and Briscoe has shown that such substances when freshly fractured have peculiar properties, owing to the fact that a "raw edge" of their atomic lattice is exposed, and having unsatisfied valencies is avid for union with anything that may be available.

The most commonly available material is water vapour, and thus freshly fractured crystalline silica becomes hydrated, and in this form it shows its highest solubility.

It is clear that the introduction of modern blasting methods and high-speed mechanical drilling in the mining industry, and of high-pressure sand-blasting in foundries, must have produced a very high content of freshly fractured crystals of silica in certain industrial dusts.

Denny[17] and his co-workers exposed a number of rabbits to silica dust constantly freshly pulverised by a machine placed in the animal chamber, and a similar number of rabbits to "stale" silica dust (not freshly pulverised). The animals exposed to the fresh dust uniformly and rapidly developed silicotic pulmonary fibrosis in five or six months, while the "stale dust" group showed only moderate pulmonary fibrosis at the end of eighteen months.

Meanwhile chemical research had demonstrated that finely powdered metallic aluminium was among the most effective "depressors" of silica solubility, and accordingly Denny[18] exposed a second series of rabbits to fresh

quartz dust, and a control series to fresh quartz dust with 1 per cent. of powdered aluminium dust.

The first batch developed silicotic fibrosis as before, but the animals receiving aluminium showed none at the end of eighteen months.

The animals protected by aluminium and those unprotected showed the same quantity of silica in their lungs; thus the inhibition of silicosis unquestionably occurred within the body.

It was found, too, that inhalation of the aluminium dust for forty minutes before or after the twelve hours' daily quartz-dusting was protective.

It has been shown that the powdered aluminium combines with the silica, satisfying its free valencies and forming an insoluble coating of gelatinous alumina.

The above work can be correlated with the fact, known for long in mining, that the admixture of other minerals with silica had some bearing on the incidence of silicosis; thus in hæmatite mining the average exposure before silicosis develops is over twenty-one years, in granite cutting it is fifteen to twenty years, in tin mining it is ten to fifteen years, and in lead, zinc, and gold-mining silicosis used to develop in three to seven years.

It is improbable that these variations are explained simply by variation in the silica content of the dust, for in precontrol days this was very high in all the above industries. The explanation probably lies in two sets of factors, one operating in the atmosphere, and affecting the settling-rate, etc., of the particles, and the other acting within the body by inhibiting solubility.

In recent years striking progress has been made at the McIntyre Group laboratory in Canada with the application of this work on aluminium inhalation to workers in the metal mines there, and while this matter does not come within the scope of a paper dealing with the ætiology of silicosis it may be stated that the recent publications of these workers and of Crombie,[19] Blaisdell, and McPherson contain great promise of the successful application of their laboratory findings to the field of human pathology and prophylaxis.

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REPORTS OF SOCIETIES

JOINT TUBERCULOSIS COUNCIL

THE Joint Tuberculosis Council, at their last meeting (Saturday, May 12, 1945), discussed several matters of interest to the tuberculosis world.

On the recommendation of the Joint Education Committee (upon which the Council and the National Association for the Prevention of Tuberculosis are represented), the Council approved the draft constitution for a Tuberculosis Educational Institute. This Institute will arrange courses, lectures and demonstrations for the education of doctors, nurses, almoners, and social workers in tuberculosis. It will, it is hoped, act as an information centre in all matters connected with tuberculosis education, and will recommend candidates for N.A.P.T. scholarships. The Institute will suggest lines of research in tuberculosis, and will generally facilitate and encourage the study of the subject in Great Britain by visitors from overseas. It will work in co-operation with university post-graduate centres and will give them all possible assistance.

The Joint Education Committee also recommended the N.A.P.T. to award two £50 scholarships for health visitors, and two scholarships of similar amount for student almoners.

The Council's Committee on Reorganisation of the Tuberculosis Services (which at this meeting was re-named the Committee on Development of the Tuberculosis Services) was asked to consider and report upon the present situation in relation to the proposed National Health Service (with particular reference to the recent negotiations between the Ministry of Health and the B.M.A.).

The Council decided to approach the Spens Committee with representations that tuberculosis clinicians should be dealt with, in any scheme of salary revision for practitioners engaged in public work, on the same basis as specialists in other branches of medicine.

THE Joint Tuberculosis Council held a Special Meeting on Saturday, July 21, 1945, at the London School of Hygiene and Tropical Medicine, to consider some important reports prepared by committees.

The Committee on Notification of Tuberculosis, which has for some time been considering whether the present method of notification really achieves the best results, proposed some important changes in the present system. The most notable is a suggestion that notification in the future be a two-stage procedure. The first stage would be that of *intimation*. A practitioner, suspecting that a patient might be tuberculous, would be able to send a non-committal intimation to the local authority instead of, as now, being compelled to make up his mind and decide upon a definite diagnosis of tuberculosis, which automatically puts the patient on the register of tuberculous persons. Under the new procedure the final diagnosis of the patient, and therefore the responsibility of placing him on the register, would rest with the tuberculosis officer. At the same time there would be nothing to prevent the general practitioner who was certain of his ground from notifying the patient exactly as is done now. The value of the suggested new system would be that provision would be made for the doubtful case, whose illness could be thoroughly investigated by a tuberculosis consultant without any possible " registration " stigma in the event of his turning out not to have the disease.

The Council decided to forward the Committee's report to the Ministry of Health and to suggest that a meeting be arranged to discuss the best methods of achieving its objects. The report will not be distributed as a J.T.C. publication until the consultations with the Ministry have taken place.

A report which the Council will shortly publish is the Interim Report of the Committee on Ministry of Health Memorandum 266/T. This report raises three different kinds of issues arising out of Memorandum 266/T. First of all there are administrative amendments, which could be authorised quite simply by an amending memorandum. Secondly, there are many points on which interpretative guidance is needed by bodies and officers administering the scheme; these could be dealt with by Ministry circulars, or guidance could be given by an authoritative body such as the Joint Tuberculosis Council itself. Thirdly, there are the matters in which information rather than specific action is necessary. Some method is urgently needed whereby administering authorities can exchange information and pool their experience.

The report points out that the financial allowances scheme during the financial year ended March 31, 1945, cost the Exchequer £650,000 as against the estimated £3,000,000 mentioned by the Minister of Health at the N.A.P.T. Conference.

The Committee believe that the basic monetary allowance under 266/T should be substantially increased and that dependents' allowances should be increased also. The report states that, excluding supplementary allowances from local authorities or care committees for extra nourishment, the maintenance allowance is less than the amount given by some Public Assistance authorities. Patients have been known to ask whether they could remain on relief rather than come under 266/T. " There is little inducement in the scale of allowances," says the report, " to persuade the early cases with few or no symptoms and earning good wages to give up their work while the disease remains early. For many patients, the anxieties of today are more potent than the fears of tomorrow, which they hope may never materialise." The Council authorised the issue of a questionnaire to local authorities with the object of collecting some data on the operation of Memorandum 266/T. The information, when obtained, will be reviewed by the Committee in a further report.

The third main subject dealt with at the Council meeting was concerned with the conversion of the hospital and sanatoria staff from Mantoux negative to Mantoux positive. It was decided to prepare a " Memorandum of Advice " on this subject, to be published shortly under the auspices of the Joint Tuberculosis Council. The main points of the memorandum will include a month's " work and bed " for staff presenting no symptoms, no radiological evidence and no significant increase in the sedimentation rate. For the symptomless with an increased sedimentation rate a month's " half-duty and rest " is suggested, and if at the end of that time the sedimentation rate is normal, full duty will be resumed. For those without symptoms, but with enlarged hilar glands and an increased sedimentation rate a month in bed is the recommendation, to be followed by a month's " half-duty and rest " if this succeeds. Those who are actually ill, of course, are treated for as long as is necessary.

It is most important, the J.T.C. believe, that Mantoux negative staff should be told that they are negative, and that they are almost certain to become positive. They should also be told that just as most town dwellers go through a primary tuberculosis infection at some time in their lives and know nothing about it, the same uneventful course is probably the lot of the sanatorium nurse or maid converting from negative to positive.

NOTICES

BRITISH LEGION VILLAGE

Preston Hall, Near Maidstone, Kent.

The Federation of the British Legion has approved the recommendation of the Council of Management of the British Legion Village that the following be appointed as Honorary Consultants to Preston Hall, Maidstone, Kent; British Legion Sanatorium, Nayland, Colchester, Essex; and Douglas House, Bournemouth, the convalescent seaside annexe of Preston Hall.

During the absence of Dr. J. B. McDougall (Medical Director of the British Legion Village), who is now attached to U.N.R.R.A., Dr. Frederick Heaf has been appointed Honorary Consulting Medical Director.

Honorary Consulting Physicians.—Surgeon-Captain W. D. W. Brooks, R.N.V.R., M.A., D.M., F.R.C.P.; E. T. D. Fletcher, Esq., M.A., M.D., M.R.C.P.; F. R. G. Heaf, Esq., M.A., M.D., M.R.C.P.; J. L. Livingstone, Esq., M.D., F.R.C.P.; H. V. Morlock, Esq., M.C., M.D., F.R.C.P.; C. Spencer Palmer, Esq., M.R.C.S., L.R.C.P.; J. Douglas Robertson, Esq., M.D., Ph.D., D.Sc., M.R.C.P., D.P.H., F.R.S.E.; Major N. Lloyd Rusby, M.A., D.M., M.R.C.P.

Honorary Consulting Surgeons.—A. T. Fripp, Esq., M.B., B.Ch., F.R.C.S.; Surgeon-Commander G. A. Mason, R.N.V.R., M.B., B.S., F.R.C.S.; Major-General P. H. Mitchiner, C.B., C.B.E., M.D., M.S., F.R.C.S., D.Ch.; E. G. Slesinger, Esq., O.B.E., M.S., F.R.C.S.

Honorary Consulting Pathologist.—Major J. E. McCartney, R.A.M.C., M.D., D.Sc.

Honorary Consulting Laryngologist.—Major R. Scott Stevenson, R.A.M.C., M.D., F.R.C.S. Ed.

Honorary Consulting Radiologists.—T. V. L. Crichlow, Esq., M.R.C.S., L.R.C.P., D.M.R.E.; Major D. B. McGrigor, M.B., Ch.B., D.M.R.E.

Honorary Consulting Gynæcologists.—J. D. Barris, Esq., B.A., M.B., F.R.C.P., F.R.C.S., F.R.C.O.G.; G. F. Gibberd, Esq., M.S., M.B., F.R.C.S., F.R.C.O.G.

Honorary Consulting Anæsthetist.—G. S. W. Organe, Esq., M.A., M.D., D.A.

Honorary Consulting Dental Surgeons.—Major-General J. P. Helliwell, C.B.E., M.R.C.S., L.R.C.P., L.D.S.; Surgeon-Commander H. Paxton Baylis, R.N.V.R., V.D., M.R.C.S., L.R.C.P., L.D.S.

The Consultants will form a Medical Committee under the Chairmanship of the Honorary Consulting Medical Director to advise the Council of Management in all medical matters connected with the British Legion Village.

TUBERCULOSIS EDUCATIONAL INSTITUTE

Tuberculosis is a great national problem, and authoritative knowledge is required nowadays not only by doctors and medical students, but also by social workers, health visitors and interested laymen engaged in the campaign against the disease.

In the near future new schemes of treatment, research and rehabilitation will increase the need for education and training in modern ideas. Overseas and Empire visitors, doctors and other health workers, require instruction in British methods and anti-tuberculosis measures. To form a central body

which can supply these needs, the N.A.P.T. and J.T.C. have formed a new organisation to be called the Tuberculosis Educational Institute. This will be managed by a Committee under the Chairmanship of Dr. Frederick Heaf, with the following members: Professor Tytler, Dr. A. S. Hall, Dr. A. Morland, Dr. Simpson, Dr. Norman Smith, Dr. D. P. Sutherland and Dr. R. A. Young.

The functions of the new body will be as follows:

(*a*) To arrange courses, lectures and demonstrations for the education of doctors, nurses, almoners and social workers in tuberculosis.
(*b*) To act as a centre of information in all matters connected with education in tuberculosis.
(*c*) To recommend candidates for N.A.P.T. scholarships to assist doctors, almoners, nurses and social workers to study tuberculosis.
(*d*) To suggest lines of research in tuberculosis.
(*e*) To issue a half-yearly index of tuberculosis literature.
(*f*) To facilitate and encourage the study of tuberculosis in this country by visitors from overseas.

The Tuberculosis Educational Institute will be of help to the individual doctor or health worker seeking knowledge on the subject.

A register of lecturers and tutors will be formed who will be available to give instructions to workers at home or from overseas.

A Managing Committee has arranged many successful Refresher Courses for doctors, health visitors and social workers, and with co-operation from post-graduate medical schools these activities will be extended as opportunity offers to all parts of the country.

The Secretary of the Institute will be the Secretary-General of the N.A.P.T. Dr. Harley Williams, who has been placed at the service of the Joint Committee for Education (since its inauguration in 1943) by the Council of the N.A.P.T.

The work of the Tuberculosis Educational Institute will be planned to fit in with the general scheme of post-graduate education now being planned.

REVIEWS OF BOOKS

Mass Miniature Radiography of Civilians. By K. C. Clark, P. D'Arcy Hart, P. Kerley and B. C. Thompson. Medical Research Council, Special Report Series No. 251. London: H.M. Stationery Office. 1945. Price 3s. 6d.

This report deals with 23,000 examinations made by the mass miniature radiography method. The results do not differ substantially from those already published by other workers, and in this way the report adds little to our knowledge of pulmonary disease. However, it does a valuable service by providing a most comprehensive account of the running of a mass miniature radiography unit. Every detail of the equipment, personnel and administration is described, and the work is full of useful hints on the use of apparatus, developing of films, viewing the miniature pictures and so forth. Some good illustrations help to clarify the text, and the work will be welcomed by those interested in the practical technique of mass radiography. The procedure recommended for dealing with observation cases leaves much to be desired, since it reflects the attitude of the administrator rather than that of the clinician.

Artificial Pneumothorax in Pulmonary Tuberculosis. By T. N. RAFFERTY. London: Wm. Heinemann (Medical Books), Ltd. 1944. Price 21s.

This new book is not intended to be a practical manual for those who have to carry out pneumothorax treatment; it is rather a theoretical study of the place of this treatment in the armamentarium of the phthisiologist. The useful bibliography reflects the trouble which the author has taken to obtain statistical evidence in support of his views, and he presents a well-reasoned study of the place of pneumothorax treatment in the collapse therapy of pulmonary tuberculosis. The indications for this treatment, its results and complications, are described in detail. Particular stress is laid upon the importance of obtaining a satisfactory collapse of the diseased area, as judged by freedom from adhesions and by the closure of cavities. The collected evidence gives incontrovertible proof of the necessity of refusing to be satisfied with anything less than a perfect collapse, and unless this can be obtained the game is not worth the candle. Whatever a patient may gain from an imperfect pneumothorax, he stands to lose far more from its complications.

A valuable chapter is devoted to the problems raised by tuberculous tracheobronchitis, a complication which has been studied more extensively in America than in this country and which has a most important bearing upon the choice of collapse measures. There is also a good account of the behaviour of tension cavities. The chapters dealing with the management of a pneumothorax and of its complications are disappointing, since they deal too much with general principles and contain little detailed guidance. Such an approach is too often reminiscent of Peacock's philosopher " who from equal measures could always produce arguments on both sides of a question, with so much nicety and exactness, as to keep the said question eternally pending, and the balance of the controversy perpetually *in statu quo*."

Nevertheless, this is a useful little book, and the excellence of the production makes it a pleasure to read.

Social Work for the Tuberculous—A Practical Guide. By HARLEY WILLIAMS and IRENE HARBERT. N.A.P.T. Price 5s.

The social problems which attend a disease are usually proportional to its chronicity, and in tuberculosis, as with rheumatic and mental conditions, the financial and domestic difficulties are often harder than the medical to solve. Much of the work in this field is left to the almoners and social workers, and it is primarily as a guide for them that the N.A.P.T. has published this excellent little book; nevertheless the information it contains is of value to everyone concerned with the care and rehabilitation of tuberculous patients, and it should find a welcome place on many a doctor's desk.

The schemes which provide financial and other assistance to patients both during and after institutional treatment are described, among them care committees, voluntary and Government organisations, village settlements and the work of the health visitor. The section on the treatment allowances payable under Memorandum 266/T is particularly clear and detailed, and contains examples of the assessment of typical cases. Perhaps one gains too rosy a view of the patient's lot—the gaps which remain in our provision for chronic and incurable cases are still great, and although the Ministry of Labour's arrangements for the retraining of the partially disabled sound promising, they do not yet cover those candidates for rehabilitation who retain a positive sputum.

One of the most valuable features of this book is the list, arranged in counties and complete with addresses, of all the organisations which can be called on to give aid in individual cases. The length of this list is an encouraging reassurance of man's humanity to man.

Hygiene. By J. R. Currie and A. G. Mearns. Livingstone, Edinburgh. Price 21s.

Tuberculosis is a community disease of such importance that one would expect it to figure largely in any work on public health. Although it receives slightly more space in this book than any other disease, the use of that space is not well handled, for there is too much clinical detail, and too little consideration of the wider problems of social, environmental and occupational factors in the occurrence and control of tuberculosis. The only figures are those showing the fall in the total death rate from all forms of the disease since 1871, and no analysis is made of them. The pneumonoconioses receive brief mention, with the method of applying for compensation, but there is no reminder of the time limit on claims made after leaving the industrial hazard. This book, which is beautifully produced and illustrated, suffers throughout from the same unbalanced selection of material that is noticeable in its treatment of pulmonary conditions, but it contains a good deal of interesting matter and is written in a concise and readable style.

Familial Susceptibility to Tuberculosis: Its Importance as a Public Health Problem. By Ruth Rice Puffer. The Harvard University Press. Price $2.00.

Of all the diseases whose causal agents are known, there are few in which the factors responsible for the development of the condition are so many and so uncertain as in tuberculosis. Even the importance of exposure to the organism is complicated by the controversy of exogenous versus endogenous reinfection, and the nature and degree of immunity conferred by the initial infection is still obscure. As belief in the actual inheritance of the disease died out, the significance of environmental, infective, and nutritional factors came into prominence, and the question of familial susceptibility received only half-hearted attention. Statistical studies of tuberculous families have, however, continued, and the resulting evidence has now been comprehensively reviewed by Ruth Rice Puffer in a most interesting and stimulating book.

That tuberculosis develops more frequently in siblings of index cases—*i.e.*, initial cases whose families are under review—than in siblings of their consorts, and that it also develops frequently in consorts of index cases, does not separate hereditary from environmental factors, but only shows that these are persons at risk. But when an analysis is made of families of consorts it is found that the proportion of husbands or wives developing the disease is higher among those with a tuberculous family history than among those without. Although this is suggestive, much more impressive results are obtained by the study of mono- and di.zygotic twins, for 66·7 per cent. of monozygotic cotwins of index cases developed tuberculosis, compared with only 23·0 per cent. of dizygotic cotwins —the latter figure being closely comparable to that for all siblings. Studies of the children of tuberculous parents and the parents of tuberculous children all point to a familial influence apart from the effects of environment, and the fact that the mortality in affected families has remained almost unchanged during decades in which the total tuberculosis mortality has declined suggests the

existence of tuberculous families which themselves are dying out, but among whom the greater majority of cases are aggregated.

The statistical results of all these investigations are ably surveyed and analysed in this book, and the conclusions drawn are considered in their relation to plans for the control of tuberculosis; the methods of analysis are explained in an appendix. This is a book which, though by no means easy to read, amply repays study and consideration; it casts weighty evidence on the side of familial susceptibility as an important factor in the development of tuberculosis, and it points the way to renewed efforts in case-finding by Public Health authorities.